subside when the catheter tip is in the pulmonary artery. Symptomatic pulmonary infarction may result if the catheter floats into a permanent wedge position or the balloon is left inflated for a prolonged time. Thrombosis associated with the pulmonary artery catheter has been reported, as has perforation of the pulmonary artery and knotting about intracardiac structures. Balloon rupture may occur if it is overinflated, or if the catheter is reused. Sterile technique during insertion and manipulation of the catheter will minimize the risk of infection.

Despite its shortcomings and complications, the flotation pulmonary artery catheter is a safe and practical clinical device that provides reasonably accurate information about left ventricular function.

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Anesthetic Management for Patients with Coronary Artery Disease

THE MOST CHALLENGING and important problem facing an anesthesiologist caring for a patient with coronary artery disease is the control of factors affecting myocardial oxygen supply and demand.

The major controllable determinant of myocardial oxygen supply is the diastolic arterial blood pressure. Below a critical lower pressure limit, reduced flow across narrowed coronary arteries may cause a loss of autoregulation of coronary flow. Therefore, anesthetists must keep patients' diastolic blood pressure high enough to ensure adequate coronary blood flow. Although critical lower pressure limits vary from patient to patient, most anesthetists attempt to maintain the diastolic pressure above 50 mm of mercury. One should suspect a problem with low myocardial oxygen supply if changes in ST segments, arrhythmias or pump failure occur.

The four major determinants of myocardial oxygen demand are heart rate, afterload (the systolic blood pressure plus any aortic valve pressure gradient), preload and the state of myocardial contractility. The rate-pressure product (heart rate times systolic blood pressure) correlates well with myocardial oxygen demand. In the presence of an increasing rate-pressure product, changes in ST segments, arrhythmias or pump failure should lead an anesthetist to suspect that myocardial oxygen demand exceeds supply. In general, high myocardial oxygen demand is produced by patient anxiety, light anesthesia (especially during intubation), lack of β-adrenergic blockade, or the overuse of pressor or cardiotonic drugs.

Adequate monitoring of patients with coronary heart disease requires a continuous electrocardiogram on which ischemic ST-segment changes can be seen. If possible, lead V5 should be monitored, since it is usually the most sensitive in evaluating changes in the ST-segment. In the absence of V lead capabilities, lead 2 is the best second choice. Direct intraarterial blood pressure monitoring is recommended so that second-to-second changes can be observed, and in addition, arterial blood-gas determinations should be made. If the disease is severe or there is substantial left ventricular dysfunction (or both), a pulmonary artery catheter (Swan-Ganz) should be placed in order to continuously determine the left ventricular filling pressures (preload).

It is important to realize that there are two major categories of patients with coronary artery disease, requiring notably different management techniques. The first includes those patients with little or no myocardial damage, a normal cardiac reserve, no history of cardiac failure, a left ventricular end-diastolic pressure (LVEDP) under 14 mm of mercury, ejection fraction over 0.4 and no left ventricular aneurysm. These patients tend to have hyperdynamic responses to any stimulus (for example, anxiety or pain). Management should include adequate preoperative discussion and sedation, adequate anesthesia, β-adrenergic blockade both preoperatively and intraoperatively, the use of intravenously given nitroglycerine or sodium nitroprusside (or both), intratracheal spray with lidocaine before tracheal intubation and possibly, metocurine instead of pancuronium. The use of enflurane or halothane, either as the major anesthetic agent, or as an adjunct to a narcotic technique, is desirable. All of the above measures tend to minimize increases in myocardial oxygen demand.

The second patient category includes patients with previous major cardiac damage, including ventricular aneurysm, mitral or aortic valvular dysfunction, heart failure or previous myocardial infarction, an LVEDP over 14 mm of mercury, an

ejection fraction under 0.4 and, therefore, a compromised cardiac reserve. In these patients heart failure tends to develop easily if myocardial depressants are used. Therefore, propranolol, halogenated anesthetic agents and thiopental must be used cautiously, if at all.

It is essential, therefore, that patients receive a thorough preoperative evaluation, and be appropriately prepared for anesthesia. The plan of anesthetic management should have as its major goal the maximizing of myocardial oxygen supply, and minimizing of myocardial oxygen consumption.

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Protection of the Brain From Progressive Ischemia

INCREASING CAPABILITY in cardiopulmonary resuscitation both in and outside hospitals has resulted in a large number of patients whose cardiovascular and pulmonary systems are stable but who have sustained severe brain damage. As a result, a considerable interest in the concepts of cerebral resuscitation has developed. In the past, varying combinations of steroids, osmotic diuresis, hypothermia and hypocarbia have not ensured recovery without neurologic deficit following severe global ischemia.

Recent studies suggest that barbiturates may have a protective effect following both focal and global ischemia. Investigators at the University of Pittsburgh have shown there to be pronounced decreases in neurologic deficits when thiopental was administered to primates following 16 minutes of cerebral ischemia. In a preliminary pilot study at Stanford University Medical Center, four patients having sustained cardiac arrests with estimated global cerebral ischemia in excess of seven to ten minutes were given sodium thiopental in doses averaging 578 mg per kg of body weight over times ranging from 48 to 72 hours. Criteria for determination of dose were achievement of blood levels between 40 and 90 μg per ml and an isoelectric electroencephalogram for 72 hours. Two of the four patients made a complete neurologic recovery. Similar beneficial neurologic recovery has been shown recently in patients with Reye syndrome, and following near-drowning or severe head trauma.

Mechanisms of action cited for the apparent success of barbiturates following cerebral ischemia include decreased cerebral edema, decreased intracranial pressure, interference with the "no re-flow" phenomenon, scavenging free chemical radicals and decreased cerebral oxygen demand. Although it is far too premature to make definitive statements as to the efficacy of barbiturates as a protective treatment for cerebral ischemia, experimental and clinical results appear quite promising. A prospective randomized study now is necessary to prove the value of this regimen. The dismal neurologic results following cardiac arrest attest to the need for developing improved diagnostic, therapeutic and monitoring techniques in the management of cerebral ischemia.

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Malignant Hyperthermia: An Update

MALIGNANT HYPERTHERMIA (MH), first described by Denborough in 1960, is a pharmacogenetic abnormality transmitted as an autosomal dominant pattern with reduced penetrance and variable expressivity. It may be triggered by inhalational anesthetics or depolarizing muscle relaxants and presents clinically as a hypermetabolic state, primarily of the skeletal muscle. Early clinical symptoms are tachycardia, tachypnea, and a respiratory and metabolic acidosis. Muscular rigidity of the jaw or extremities may or may not occur in the syndrome. Profuse skin sweating and ventricular arrhythmias have also been commonly described, with an increase in body temperature appearing as a later sign. Not all cases of MH